

TUBERCULOSIS OF THE SPLEEN, SEPTIC INFARCTION, POLYCYTHEMIA, SPLENECTOMY.

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SECONDARY tuberculosis of the spleen is not uncommon. True primary tuberculosis of the spleen is probably extremely rare. In the cases of so-called primary tuberculosis of the spleen described in the literature the tuberculous process in the spleen forms the main lesion, the probable original focus or point of entry, in the lungs or lymph nodes, for example, remaining quiescent or becoming healed, while the process in the spleen develops, and is the cause of the symptoms presented by the patient.

The first case of primary tuberculosis of the spleen was reported from autopsy by Coley¹ in 1846. The first reported splenectomy for splenic tuberculosis is that of Burke² in 1889. The patient died the following day, apparently from sepsis. No autopsy was performed in this case, but previous physical and sputum examination showed no evidence of pulmonary tuberculosis. In 1898 Quenu and Baudet³ collected and reported five cases, including one of their own. In 1899 Rendu and Vidal⁴ described a symptom-complex including tuberculosis of the spleen, polycythemia without leukemia, and cyanosis. They attributed the symptom-complex to the splenic lesion. Bayer⁵ collected, up to 1900, 9 operations with 7 recoveries, including 1 of his own, and 19 autopsy cases. He emphasizes the importance of splenectomy, and also the relative diagnostic importance of polycythemia and cyanosis. His patient had 6,000,000 red cells, 7500 white cells, hemoglobin 40 per cent., and was pale. G. B. Johnson⁶ in an article on splenectomy published in 1908 included reports of ten cases of splenectomy for tuberculosis, with two deaths.

The most recent and complete article on this subject is that by Winternitz,⁷ who has collected and tabulated 51 cases from the literature; 16 of these cases have been operated on, 9 cases recovered (56 per cent.). The list includes 3 cases; 1 by Ciaccio,⁸ in which

¹ Trans. Path. Soc., London, 1846, i, 276.

² Dublin Jour. Med. Sci., 1889, xlvii, 540.

³ Rev. de gynéc. et de chir. abd., 1898, ii, 317.

⁴ Bull. et mém. Soc. de Med. des hôp., 1899, Series 3, xvi, 528.

⁵ Mitt. a. d. Grenzgeb. d. med. u. chir., 1904, xlii, 523.

⁶ Ann. of Surg., 1908, xlviii, 50.

⁷ Archiv Int. Med., 1912, ix, 680.

⁸ Deutsch. Zeitsch. f. Chir., 1909, lviii, 404.

splenectomy was not done on account of adhesions; 1 by Hayden,⁹ in which the spleen was sewed to the abdominal wall and drained; and 1 by Hamilton,¹⁰ in which the same procedure was carried out. His analysis and summary of the 51 cases is so thorough and complete that the conclusions found as a result of his study may be given here as follows:

Tuberculosis of the spleen seems to be equally frequent in the male and female sex. It is most frequent between the ages of twenty and forty, but may occur at any age, the youngest in his tabulated list being one year, the oldest eighty years. The symptoms may run an acute or chronic course, in the acute cases the symptoms were those of an acute systemic infection, and in the chronic cases the onset was characterized by pain or tumor in the splenic region (70 per cent. of cases). There may be, in addition, gastro-intestinal or respiratory disturbances. In the list of 51 reported cases the blood count was given in 26. It was normal in 9 (34.61 per cent.), and showed anemia in 11 (42.30 per cent.); polycythemia was present in 6 (23.08 per cent.). The anemia was not marked in any instance, 3,200,000 being the lowest count. As a rule the leukocytes were not increased, and their number seems totally independent of the number of red cells. In his conclusion, Winternitz states that "death invariably follows if the spleen is not removed."

In contrast to this statement of Winternitz we have the statement of Osler¹¹ in an article on Erythrocythemia, "Splenectomy should never be done in these cases." And the list of reported cases in which the blood picture shows a true polycythemia bears out this latter conclusion. In view of the fact that in our case tuberculosis of the spleen and polycythemia co-existed, these conflicting statements make the question of the treatment of such cases a difficult one. Owing to the apparent relationship between tuberculosis of the spleen and polycythemia it may be of interest in this place to consider briefly what is known of polycythemia, excluding those cases due to change in altitude or chronic coal-tar products poisoning:

Polycythemia as a definite disease was first described by Vaques¹² in 1892. Osler¹³ in 1903 and Turk¹⁴ in 1904 brought the syndrome into general notice (enlarged spleen, cyanosis, and high red-count), with early case reports by McKeen¹⁵ and Saundby and Russell.¹⁶ Since then so many cases have been reported in the literature that in 1911 Watson Weimys,¹⁷ in a review of the subject, cites sixty references.

⁹ Trans. Indiana State Med. Soc., 1897, xlviii, 96.

¹⁰ Internat. Clin., 1896, vi, Series 3, 213.

¹¹ Principles and Practice of Medicine, 1912, Eighth edition, 767.

¹² Compt. rend. Soc. de biol., Paris, 1892, xlv, 384.

¹³ AMER. JOUR. MED. SCI., 1903, cxxvi, 187.

¹⁴ Boston Med. and Surg. Jour., 1901, 144.

¹⁵ Edinburgh Med. Jour., 1911, vol. vi.

¹⁶ Wien. klin. Woch., 1904, xvi, 153.

¹⁷ Lancet, 1901, i, 515.

There are five theories as to the etiology of polycythemia: (1) That the disease is a primary myelopathy (Parks Weber),¹⁸ and Jacobs¹⁹ writes of the disease as a neoplasm of the blood similar to a neoplasm of the tissues in cancer; (2) splenic tuberculosis (Rendu and Vidal²⁰ and Lefas);²¹ (3) diminished destructibility of the red blood corpuscles; (4) diminished oxygen-carrying power of the hemoglobin (Bence);²² (5) theory of blood-stasis, the extreme viscosity of the blood being a marked phenomenon in these cases of polycythemia.

The above brief review of the literature on the subject is enough to indicate how little really has been definitely determined as to the etiology and essential pathology of polycythemia. Moreover, the direct relation between polycythemia and tuberculosis of the spleen is open to question. Therefore, while the multiplicity of lesions demonstrated by autopsy, in the following case, prevents definite conclusions, the history, report of operation, clinical data, and report of autopsy are presented as a contribution to the literature on the subject.

For much of the previous history of this patient, and for the clinical data extending over a period of seven years, we are indebted to Dr. J. A. Travell, of New York.

CASE I.—J. R. S., aged thirty-nine years; salesman; born in United States.

Family History. Mother died of nephritis, otherwise irrelevant.

Previous History. Had chills and fever when a child; in youth had frequent severe nose-bleed, but in 1901 an operation for abscess in the inguinal region was not accompanied by unusual bleeding. In 1902 had pneumonia, followed by pleurisy. Was formerly a hard drinker, but later a total abstainer. Had always been constipated. Normal weight was about 180 pounds.

History taken in 1905 stated that there had been no recent cough, catarrh, or pleurisy. During the previous year he had begun to have frequent attacks, in which his face turned purple, and he became nervous, but never lost consciousness. At that time he was recovering from an attack of nervous prostration, and was troubled with indigestion and spots which flickered before his eyes. He, however, had no headache. Weight was then 147½ pounds. Physical examination showed right kidney palpable; heart lungs, and urine normal; face flushed. Intense congestion of all mucous membranes, but legs and body pale.

January, 1907. For a period of three weeks had had momentary attacks of complete blindness of the right eye from one to six times a day. An oculist reported the eyes were normal and that the

¹⁸ Quarterly Jour. Med., 1908, vol. ii.

¹⁹ Münch. med. Woch., 1912, lix, No. 41.

²¹ Loc. cit.

²² Deutsch. med. Woch., 1906, xxxii, 1451.

²⁰ Paris Thesis, 1903.

trouble was due to vasomotor disturbance. All capillaries of the body were intensely congested. Was always constipated.

April 6, 1908. Extreme capillary congestion of the face and mucous membrane of the mouth. Color below the neck was normal; feet were cool. Had epigastric pain at times, with gas in the stomach. Heart was normal; radial and temporal arteries were hard. Spleen was palpable one inch below ribs on deep inspiration.

April 8. A gall-stone was passed after severe pain for twenty-four hours. Stone was not recovered. Hemoglobin, 90 per cent.; polynuclear leukocytes, 88 per cent.; transition leukocytes, 1 per cent.; eosinophiles, 2 per cent.; small lymphocytes, 5 per cent.; large lymphocytes, 4 per cent.

April 22. Face was flushed as usual. Abdomen was not sensitive. Liver dulness was normal. Spleen was not palpable.

May. Face was scarlet. Spleen was barely palpable below the ribs. No hepatic tenderness; no gastric splashing. Drinking buttermilk invariably caused blindness and dizziness in half an hour, the attack lasting twenty minutes.

June. Face was constantly flushed; weight, 146½ pounds. Spleen not palpable; heart, lungs, and abdomen were negative. Reflexes were present; blood-pressure, 140 mm.

July. Spleen not palpable, lacked ability to concentrate attention on work. Face was scarlet.

January, 1909. Had been well, but had frequent lapses of memory, and the face flushed annoyingly.

February. Urine examination showed albumin, acid; no sugar; granular and hyaline casts; many pus cells and spermatozoa. Quantity, two quarts in twenty-four hours. Pulse, 93 and regular, blood-pressure, 130 mm. Marked gastric splashing.

March. Had severe hemorrhage; about one quart from the bowels, immediately followed by about one quart from the stomach; both clotted heavily. Collapse; pulseless; soreness at lower left border of the stomach. The following day pulse was regular, of fair quality. No further hemorrhage. Urine, ninety-six ounces in twenty-four hours. No sugar, no albumin, many casts. Two days later the blood-pressure was 140 mm.

April. Frequent neuralgic pains in superficial vessels of the calves of the legs, lasting about five minutes. Veins distended. Face flushed, and dizzy at times. Blood-pressure, 110 mm. Weight, 145½ pounds.

May. Felt well; passed about three quarts of urine in twenty-four hours.

August. Face and hands flushed and cyanotic. Blood-pressure, 130 mm. Gastric splashing. Often lost control of himself. Slept poorly.

October. Had felt unusually well for some time. Blood-pressure,

135 mm.; weight, 152½ pounds. When badly constipated had spells, during which he momentarily lost consciousness.

June, 1910. Departed for the West. Had a good year and, as a rule, felt well. Blood-pressure, 140 mm. Heart and lungs were negative. No aneurysm. Urine showed trace of albumin; much indican. Not any blind spells.

October. While in Portland, Oregon, the patient had another severe hemorrhage, both from the stomach and bowels, and came under the care of Drs. J. O. E. King and Jones, of Portland. After this attack a careful clinical examination was made of stomach contents, stools, and blood. The gastric analysis and stool examination were negative. Blood-pressure was between 100 and 120. Leukocytes, 32,000; red cells, 6,000,000. Weight, 148 pounds. During the interval between this attack and the year 1912 he remained in the West and did no work on account of his health. He was bled once, with relief of his symptoms. While in the Battle Creek Sanatorium during this time his blood examination was red cells, 6,550,000; hemoglobin, 92 per cent.; leukocytes, 41,000. Percentage of polynuclear cells was not given. His blood-pressure was then about 120.

In the spring of 1912 he returned to New York and resumed his occupation, and reported himself in fairly good health. Six weeks before his admission to St. Luke's Hospital, November 27, 1912, he had been suffering with indigestion and some pain in the region of the spleen. He stated that eight days before, while on a business trip, after a ride of thirty miles in a wagon on two successive days over rough roads, this pain became more severe, extending to the epigastrium. Everything he ate caused excruciating pain. He did not vomit, but felt nauseated. For several days his bowels did not move. He was then seen by Dr. Egbert Le Fevre, of New York. His chief complaints at this time were severe pain in the left hypochondriac region, radiating toward the midline and across the abdomen, extreme weakness, and a feeling of fullness in the head. His temperature was 101°.

Physical Examination. The physical examination of the lungs was negative. There was an indistinct systolic murmur at the apex. Pulse in the right radial only slightly above normal in rate. Pulse in left radial absent. The spleen was palpable about three inches below the free border of the ribs, and extremely tender. A blood-count showed between 30,000 and 40,000 leukocytes, with 92 per cent. of polynuclear cells. Two days later he was seen in consultation by one of us, his condition having remained the same, except for an increased elevation of temperature and extreme difficulty in moving the bowels (only once in three days). He was then referred to St. Luke's Hospital for observation. His temperature on admission was 103°; pulse, 100; respiration, 26. His leukocytes were 23,000; polynuclear cells, 92 per cent.

At 10 P.M. on the night of admission he had a severe chill, and one hour later his temperature was 105.3°; pulse, 148; respiration, 34. Leukocytes, 39,000; polynuclears, 95 per cent. His urine showed albumin, leukocytes, and a few red cells. Believing that he had an abscess or some septic process in the spleen, an immediate operation was advised.

Operation. An incision about 20 cm. long was made along the outer border of the left rectus muscle, and the spleen was found studded with small white areas which looked like tubercles or miliary abscesses; there was also a large softened area, evidently an infarction. The spleen was only moderately adherent, and no tubercles were seen elsewhere in the peritoneum. The liver was apparently normal. A splenectomy was done with little difficulty. The lower end of the wound was closed, the upper part being left open to allow the packing and drainage with a Mikulicz drain of the upper part of the space left by removal of the spleen, from which there was a moderate amount of oozing caused by separation of the adhesions.

For the first four days following the operation the patient's temperature averaged from 100.2 to 101.4; pulse, 116 to 128; respiration, 32 to 48. Urine showed albumin and casts. Blood picture: Red blood cells, 8,800,000; hemoglobin, 120 per cent.; white blood cells, 80,000; polynuclears, 96 per cent. A few normoblasts were found, and also aniso- and poikilocytosis and polychromatophilia.

On the fifth day the patient appeared better. Temperature, 100° to 102.2°; pulse, 116 to 128; respiration, 30 to 36; red blood cells, 7,000,000; hemoglobin, 120 per cent.; white blood cells, 21,300; polynuclears, 92 per cent.; three normoblasts in two hundred cells. Bowels moved twice.

On the sixth and seventh days the general condition about the same. Blood pressure, 117. Wound dressed, and appeared in good condition. Packing removed and a rubber dam drain inserted.

On the eighth day the temperature in the morning was 100.4°; pulse, 116; blood-pressure, 118; red blood cells, 7,600,000; hemoglobin, 120+ per cent.; white blood cells, 18,000; polynuclears, 96 per cent.; five normoblasts in one hundred cells. In the evening the patient complained of abdominal pain and felt chilly. At midnight the temperature was 100.4°; pulse, 148.

December 6, the ninth day after operation, patient's condition was worse. Temperature, 101.4°. Complained of severe abdominal pain; vomited at noon. Red blood cells, 7,400,000; hemoglobin, 120 per cent; white blood cells, 23,000; polynuclears, 88 per cent. In the evening he rapidly became worse; his pulse became almost imperceptible. Rallied after intravenous infusion of saline solution. Asked for bed-pan; bowels moved. Two hours later pulse again rapidly became worse, and the patient died.

This long history is given in detail because of the number of curious facts that may be to some extent explained by the autopsy findings.

Autopsy No. 941. Because of the length of the article the autopsy findings are given in abridged form, with the omission of portions that do not seem to have a direct bearing upon the case.

Body of a poorly nourished adult male. In addition to the usual postmortem lividity there were irregular areas of dark, purplish color over the limbs and trunk, and also on the ears and cheeks. Abdomen considerably distended. Operative incision as previously described. Small amount of brownish, foul-smelling fluid free in the peritoneal cavity. Parietal peritoneum showed glistening surface, except in left hypochondrium, where the diaphragmatic and parietal surface over a region corresponding to the previous location of the spleen was thickly studded with small, raised, grayish-white nodules. The spleen had been removed at operation. The omentum was loosely adherent at the line of incision and around the pedicle of the spleen. Its color was dark and hemorrhagic. Vessels were considerably dilated. No thrombosis found.

Intestines: The intestines were markedly distended and the walls were completely relaxed. Serosa's surfaces had lost all glistening appearance. Color was dark reddish brown throughout the entire extent. Musculature and mucosa were softened and necrotic. Tissues of mesentery of the same dark color and quite swollen. No signs of obstruction or thrombosis in the vessels in the mesentery. The necrosis involved the entire intestinal tract except the sigmoid and rectum, but it was further advanced along the regions supplied by the superior mesenteric artery. The retroperitoneal glands were slightly enlarged and dark in color.

Stomach: Externally the organ showed a similar dark brownish color, but its musculature was not necrotic. The mucosa at the pyloric and cardiac extremities appeared congested, and showed some ecchymotic spots, while a band of softening about 12 cm. in width, with loss of mucosa in small areas, extended completely around the middle of the organ. No thrombosis found in gastric vessels. The organ was considerably dilated, and contained a large amount of brownish, foul-smelling fluid.

Pancreas: The surrounding and interlobular tissues were edematous, and of a dark brown color. The tail bore the structures ligated with the pedicle of the spleen. There was no evidence of hemorrhage nor of thrombosis of these vessels.

Liver: Color was dark reddish, mottled irregularly by lighter yellowish areas. Capsule was thin and smooth, except for some fibrin flakes along the left anterior margin. It measured 25 x 23 x 8 cm. Weight, 2610 gm. On section the consistency was about normal, and the cut surface presented the same yellowish, mottled appearance. A few small whitish-gray nodules resembling tubercles were found.

Kidneys: Except for their dark congested appearance and some clouding of the parenchyma the kidneys appeared normal.

Pericardium: The pericardium contained a slight excess of clear, yellowish fluid. Surface everywhere was smooth and glistening.

Heart: Subpericardial fat was small in amount. The organ appeared somewhat larger than normal. The right ventricle and auricle were distended. The apex of the left ventricle appeared rather fibrous and the color was paler than elsewhere. The right auricle and ventricle contained rather firm postmortem clots. Tricuspid valve admitted three fingers. Leaflets were smooth. The wall of the ventricle was 0.4 cm. thick. The mitral valve admitted two fingers easily. The leaflets appeared normal. The apex was occupied by what appeared from the interior as a large

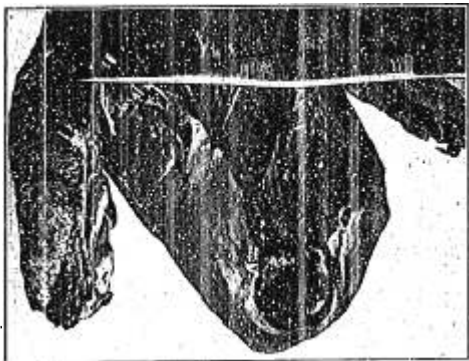


FIG. 1.—Lesions at apex of left ventricle.

mural thrombus, but which on section was evidently a subendocardial process of considerable duration. The muscle was thinned out to about 2 mm. in thickness over it, and internal to this was a dense whitish fibrous stratum about 2 mm. in thickness. Internal to this was a mass of what appeared to be softened thrombus formation. The endocardium from the lateral surface of the cavity appeared to have extended over this mass for a short distance. The diameter of this lesion was about 2.5 cm. At the point of junction of the left posterior and the anterior aortic leaflets was a fresh vegetation, with a narrow pedicle about 3 mm. long. The coronary arteries showed slight thickening and occasional patches of yellow atheroma.

Lungs: Both lungs were considerably congested, but fairly well aerated. Pleural surfaces were normal, except at the right apex

posteriorly, where a few fresh fibrinous adhesions were found. On section the upper lobe showed many conglomerate tubercles toward the central part of the lobe. These showed beginning caseation. A few rather large discrete tubercles were found in the upper right lobe. Apparently the process was a fairly recent one. The bronchial mucosa was slightly inflamed throughout, and a small amount of mucopurulent exudate was present in the larger branches. Bronchial lymph nodes considerably enlarged and showed areas of caseation.

Arteries: At the base the aorta showed numerous areas of well-advanced atheromatous degeneration without calcification or ulceration. The process was marked around the orifices of the

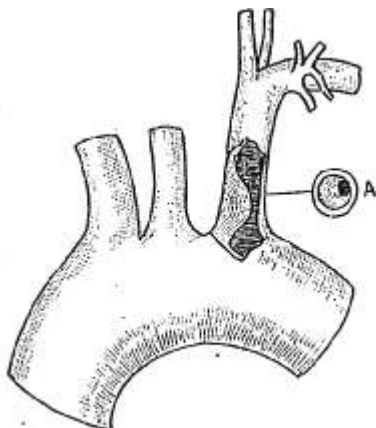


FIG. 2.—Lesion in left subclavian artery. A, Cross section of artery at point indicated.

innominate and left carotid. At the base of the subclavian artery, and extending upward for 1 cm., there was a concentric thickening of the wall, particularly on the inner anterior portion, that narrowed the lumen down to less than 2 mm. The process appeared to have been intramural rather than a canalized thrombus. The descending and thoracic portions of the aorta showed numerous plaques of yellow atheroma without ulceration or calcification, and there was a marked increase in the thickness of the arterial wall in these areas. On the posterior wall of the abdominal aorta there was an ulcerated, calcareous plaque about 1.5 cm. in diameter, to which a firm thrombus was attached. This thrombus extended into the orifice of the superior mesenteric artery, and its upper extremity

was in such a position that it could overlie the orifice of the celiac axis. The renal arteries were unaffected. The atheromatous process was present in the form of scattered plaques well down into the iliac arteries. At the bifurcation there was a firm "straddling" thrombus that extended into the iliacs for about 1 cm.

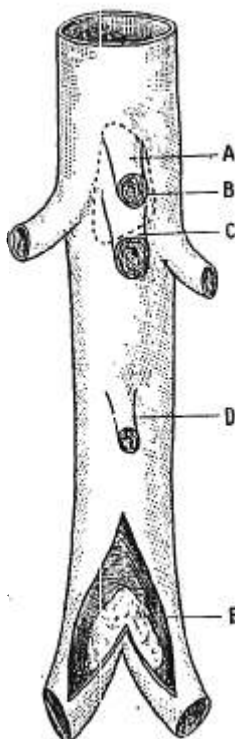


FIG. 3. — Lesions in abdominal aorta; A, obliterated lumen of celiac axis; B, location of thrombus on posterior wall of aorta; C, partial obliteration of lumen of superior mesenteric artery; D, partial obliteration of lumen of inferior mesenteric artery; E, thrombus at bifurcation of aorta.

The superior mesenteric artery was considerably sclerosed at its base, and about 0.7 cm. above this the wall was concentrically thickened so that the lumen was constricted to a diameter of 1 mm. The process appeared to be a fibrosis. Beyond this point the lumen was of normal diameter and the various branches were unobstructed. A similar, but less marked, process was present in the inferior mesenteric artery. At the beginning of the celiac axis the fibrosis was complete, and no lumen was made out. These lesions were found after removal of the organs, and it was not possible to ascertain the nature or extent of any collateral circulation that may have existed.

On opening the femur the bone-marrow was found to be of a dark grayish-red color and of soft consistency.

Anatomical Diagnosis. Gangrene of the intestines with peritonitis; thrombosis of the aorta, superior mesenteric and common iliac arteries; obstructive arteritis of celiac axis, superior and inferior mesenteric and left subclavian arteries; tuberculous peritonitis; pulmonary tuberculosis; tuberculosis of the liver; parenchymatous degeneration of the liver and kidneys; endocarditis, acute vegetative; atheroma of arteries; hyperplasia of bone-marrow. Smears from the lungs and bronchial glands showed a few tubercle bacilli.

Material from the lesion in the apex of the heart inoculated into guinea-

pigs produced typical tuberculous lesions from which tubercle bacilli were recovered.

REPORT ON THE SPLEEN. Diagnosis: Tuberculosis and Infarction of the Spleen. *Macroscopic Examination:* Specimen consisted

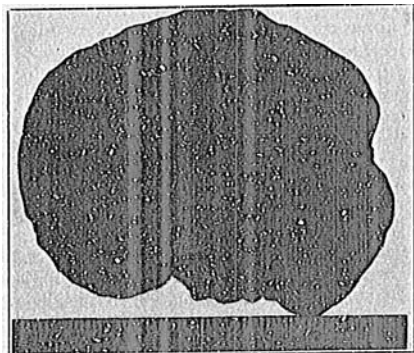


FIG. 4.—Spleen, after hardening.

of a spleen, weight 1350 grams, 22 x 14 x 9 cm. Surface was rough and nodular, with many small (1 to 3 mm.) yellowish areas scattered over the entire spleen. Capsule was thickened, and showed numer-

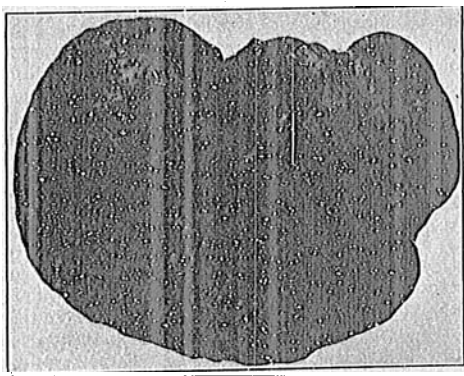


FIG. 5.—Horizontal section of spleen, after hardening, showing areas of tuberculosis and wedge-shaped infarction.

ous adhesions. Opposite hilum is an irregular area 6 x 8 cm. in diameter, much softened, over which the capsule was broken.

Cut surface was red, with innumerable yellowish areas as on the surface; these were firm and fibrous, not caseous. Soft surface area corresponded to triangular infarct extending in 8 cm.; soft fibrous, no yellowish areas. Shades off gradually through hemorrhagic zone into rest of splenic tissue.

Microscopic Examination: Small yellowish areas seen in gross are necrotic tissue, about these were zones of epithelioid proliferation and numerous giant cells of Langhans type. Fibrous tissue of spleen considerably increased. Tissue in infarcted area was entirely necrotic. Small areas of pigmentation were scattered about. No tubercle bacilli were demonstrated in section.

Microscopic Examination. Lungs: The lungs showed marked congestion of the interstitial tissue, and in some areas the alveoli were filled with red blood cells. Small discreet tubercles were numerous; many had coalesced and showed necrosis and caseation. Giant cells were particularly numerous. The smaller bronchi showed slight swelling and desquamation of their epithelium.

Bronchial Glands: Showed typical tubercle formation, with numerous giant cells of the Langhans type. Caseation was only slightly advanced. The proliferation of cells of the epithelioid type was marked. Anthracosis was present in moderate degree. The vessels were congested.

Liver: Showed pronounced congestion and a diffuse degeneration of parenchymatous cells. Many cells of endothelial type were deeply pigmented. Areas showing extravasation of blood and necrosis of the liver cells were numerous. Scattered throughout were numerous tubercles, some of which were undergoing caseation.

Kidneys: There was a moderate degree of cloudy swelling of the tubular epithelium. The glomerular tufts showed congestion and swelling of the epithelium. There was no exudate, and the capsules were not thickened. A few glomeruli were completely fibrosed, and there were a few scattered areas of interstitial fibrosis. The vessels appeared to be of normal thickness.

Diaphragm: The inferior surface in the region of the spleen showed a diffuse tuberculous process. Giant cells were numerous. A few typical tubercles were present.

Heart: Section from lesion in apex of heart showed a slight edema beneath the epicardium and a marked thickening of the walls of the arteries in this region and in the underlying muscularis. There was a slight increase of connective tissue between the muscle fibres, which were also separated by edema. About one-half of the normal thickness of the myocardium was replaced by a firm, almost hyaline fibrous tissue that had a laminated appearance and contained few nuclei. Between the muscle and this fibrous layer was a narrow zone containing many small round cells. Internal to the fibrous zone there was a mass of soft red thrombus with no evidence of organization present. In a few areas small groups

of polynuclear leukocytes were found. In one portion of the wall of the lesion there was an arrangement of the fibrous tissue in whorls that was suggestive of the fibrosis of an old tuberculous area, otherwise there was no evidence that the lesion was due to tuberculosis.

Arteries: Sections through the constricted portion of the superior mesenteric and left subclavian arteries showed a subintimal fibrous tissue hyperplasia that by concentrically thickening the wall of the vessels had markedly encroached on the lumen. In the section from the subclavian artery there was a well-marked round-cell infiltration between the area of fibrosis and the musculature, but there were no giant cells or tubercle formation.

Bone-marrow: Smears from the marrow of the femur showed the usual forms of marrow cells and an abundance of normoblasts. Normal red blood cells composed about one-third of the smear. There was practically no fat present.

A review of the history of this case for the purpose of ascertaining the sequence and possible etiology of lesions gives but little aid. From a clinical standpoint the existence of the marked polycythemia was the most interesting feature of the case. The tuberculous lesion in the spleen would seem to offer a simple explanation for this, and, in fact, attention was called to an enlargement of this organ as early as 1908, although it was not until October, 1910, that a polycythemia was demonstrated. The symptoms of flushing and congestion of the face, which continued intermittently throughout and which may or may not have had a possible cause in the polycythemia, began three years before, in 1905. Moreover, repeated examinations of the heart and lungs from 1905 until the final illness failed to reveal either a circulatory or pulmonary lesion, nor was any other evidence of tuberculosis noted.

Since primary tuberculosis of the spleen is rare, it would be reasonable to expect that some evidence of a more probable primary focus in the lungs would have been manifested. The history of pneumonia with pleurisy in 1902 is suggestive as a possible starting-point for a pulmonary focus, but the autopsy did not seem to indicate a pulmonary lesion of such long standing. However, if one wishes to regard the marked diffuse tuberculosis of the spleen as the cause of the polycythemia, a sufficiently reasonable sequence of events is present on which to base an assumption of such an etiology.

Many of the symptoms referable to disturbance of the digestive tract are readily explained, if it be agreed that the lesions found in the walls of the arteries were of ten years' duration. The various attacks of "indigestion," dizziness, blindness, and the two attacks of hemorrhage could be regarded as caused by an interference with the blood-supply of the digestive organs. In spite of the fact that the second hemorrhage was attributed to high blood-pressure,

and that venesection apparently relieved certain other symptoms, there is no record of a pressure above 140 mm. The vascular lesions must certainly have been of long duration and of a development sufficiently slow to permit of the formation of an extensive collateral circulation. The completeness of the obstruction of the celiac axis and mesenteric arteries permits of no other supposition. The lesions themselves offer little suggestion as to their origin, or to any etiological association with the polycythemia. The lesion in the apex of the left ventricle appeared to be of the same general nature and age as those in the arteries.

Whether the final attack beginning in October, 1912, was precipitated by a lighting up of an old pulmonary tuberculosis, or whether the failing blood-supply to the spleen caused a dissemination of its tubercle bacilli and a starting-point for an acute infectious process, must be left to conjecture. The most obvious cause of death was the gangrene of the intestinal tract, which must be attributed to the lesions found in its arterial supply.

Obviously there is little possibility of any inferences being drawn as to the result of operation in cases of tuberculosis of the spleen or of polycythemia, from our experience in this case. Of course, the autopsy findings demonstrating the obliterative arteritis of the celiac axis and superior mesenteric vessels resulting in gangrene of the intestines and causing death showed plainly the futility of any therapeutic measures in this particular case. And if this arteritis had not caused an early fatal termination, the lesion in the apex of the heart with its thrombus, shown to contain tubercle bacilli by guinea-pig injection, might reasonably be expected to result fatally within a short time.

It is of interest to note that in the present case, during the nine days after splenectomy, the red cell count remained high, between 8,800,000 immediately after operation to 7,400,000 on the day of his death, while the hemoglobin remained at over 120 per cent. This result one would expect if it is true that "the spleen is the graveyard of the red blood corpuscle" in the adult, although Pearce and Austin,²³ in a study of splenectomized animals, found results indicating the power of the lymph glands and liver cells to compensate, after the loss of the spleen, by taking up its function of destroying red corpuscles. However, Musser²⁴ found "that there was, after experimental splenectomy, a secondary anemia which lasted about two and one-half months." He also found a post-operative leukocytosis most marked twenty-four hours after operation, and lasting a variable time. His counts were continued one hundred and thirty-eight days in some cases, and the leukocytosis was still present. There was a marked increase in the poly-

²³ Jour. Exper. Med., December, 1912, vol. xiv.

²⁴ Archiv Int. Med., 1912, ix, 599.

nuclear cells, and a diminished lymphocyte count. In the present case this postoperative leukocytosis was a marked feature, reaching 87,000, with 98 per cent. of polynuclear cells on the second day after operation, and then gradually falling, but not back to normal.

It has previously been suggested that the tuberculosis of the spleen, in view of the autopsy findings, might be the simplest explanation for the polycythemia on the basis that the diseased spleen failed to destroy the red corpuscles. Other case reports of polycythemia, however, show numerous examples of the disease in which the spleen while enlarged was not tuberculous, and in twenty-six of the cases of tuberculosis of the spleen where the blood count was reported, collected by Winternitz, polycythemia was present only in six. Also, the condition of the bone-marrow in this and in other cases, and the blood-picture after splenectomy, would seem to indicate as a more logical presumption, that whatever may be its cause, the polycythemia, and in this case it was a polycythemia and not an erythrocythemia alone, had its direct pathological factor in the overproduction or overfunctioning of the bone-marrow. If this presumption is correct, then we can explain the enlargement of the spleen as an hypertrophy due to an effort to destroy the over-production of red cells. Finally, if there is a focus of tuberculosis elsewhere in the body from which the tubercle bacilli might enter the blood-stream, it would find in the spleen a place of diminished resistance. Thus, rather than as an etiological factor, the tuberculous spleen would be a result of the polycythemia.

Finally, while we may agree with Winternitz that in cases of tuberculosis of the spleen, when other tuberculous foci are not demonstrable, splenectomy may effect a cure; although our case proves nothing, it does add some evidence to the statement of Osler that splenectomy is contraindicated in erythrocythemia, and that when the two conditions coexist operative procedure can be of no value.

GIANT TUMORS OF THE CONUS AND CAUDA EQUINA.¹

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TUMORS of the cauda equina and of the conus cause symptoms which are considered fairly pathognomic. Nevertheless, the lesion

¹ Read at the meeting of the American Neurological Association, Washington, May 5, 1913.